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'EVENTLINE' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
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FULL ESTIMATED COST	0.21	0.21

FILE 'AGRICOLA' ENTERED AT 12:25:57 ON 18 MAY 2006

FILE 'BIOTECHNO' ENTERED AT 12:25:57 ON 18 MAY 2006

FILE 'CONFSCI' ENTERED AT 12:25:57 ON 18 MAY 2006

FILE 'HEALSAFE' ENTERED AT 12:25:57 ON 18 MAY 2006

FILE 'IMSDRUGCONF' ENTERED AT 12:25:57 ON 18 MAY 2006

FILE 'LIFESCI' ENTERED AT 12:25:57 ON 18 MAY 2006

FILE 'PASCAL' ENTERED AT 12:25:57 ON 18 MAY 2006

=> (lead or mercury) and antibody and (autism or autistic)

L1	0	FILE AGRICOLA
L2	0	FILE BIOTECHNO
L3	0	FILE CONFSCI
L4	0	FILE HEALSAFE
L5	0	FILE IMSDRUGCONF
L6	2	FILE LIFESCI
L7	0	FILE PASCAL

TOTAL FOR ALL FILES
L8 2 (LEAD OR MERCURY) AND ANTIBODY AND (AUTISM OR AUTISTIC)

=> d l8 ibib abs total

L8 ANSWER 1 OF 2 LIFESCI COPYRIGHT 2006 CSA on STN

ACCESSION NUMBER: 2005:56113 LIFESCI
TITLE: Detection of Antinuclear and Antilaminin **Antibodies** in **Autistic** Children Who Received Thimerosal-Containing Vaccines
AUTHOR: Singh, V.K.; Rivas, W.H.
CORPORATE SOURCE: Biotechnology Center Building, Utah State University, UMC 4700, Logan, UT 84322 (USA); E-mail: singhvk@cc.usu.edu
SOURCE: Journal of Biomedical Science [J. Biomed. Sci.], (20041000) vol. 11, no. 5, pp. 607-610.
ISSN: 1021-7770.
DOCUMENT TYPE: Journal
FILE SEGMENT: X
LANGUAGE: English
SUMMARY LANGUAGE: English

AB **Autism**, a neurodevelopmental disorder, may involve autoimmune pathogenesis. Since **mercury** is potentially a risk factor for autoimmunity, we conducted a study of **mercury**-induced antinuclear and antilaminin **antibodies** in **autistic** and normal children who had been pre-administered with thimerosal-containing vaccines. Laboratory analysis by different immunoassays showed that the serum level of these two autoimmune markers did not significantly differ between **autistic** and normal children. This finding suggests that the **mercury** as in thimerosal-containing vaccines is likely not related to autoimmune phenomenon in **autism**.

L8 ANSWER 2 OF 2 LIFESCI COPYRIGHT 2006 CSA on STN
ACCESSION NUMBER: 2004:108019 LIFESCI
TITLE: Infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes are major instigators of autoimmunity in **autism**
AUTHOR: Vojdani, A.; Pangborn, J.B.; Vojdani, E.; Cooper, E.L.
CORPORATE SOURCE: 8693 Wilshire Blvd., Ste. 200, Beverly Hills, CA 90211, USA; E-mail: DrAri@msn.com
SOURCE: International Journal of Immunopathology and Pharmacology [Int. J. Immunopathol. Pharmacol.], (20031200) vol. 16, no. 3, pp. 189-199.
ISSN: 0394-6320.
DOCUMENT TYPE: Journal
FILE SEGMENT: F
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Similar to many complex autoimmune diseases, genetic and environmental factors including diet, infection and xenobiotics play a critical role in the development of **autism**. In this study, we postulated that infectious agent antigens such as streptokinase, dietary peptides (gliadin and casein) and ethyl **mercury** (xenobiotic) bind to different lymphocyte receptors and tissue enzyme (DPP IV or CD26). We assessed this hypothesis first by measuring IgG, IgM and IgA **antibodies** against CD26, CD69, streptokinase (SK), gliadin and casein peptides and against ethyl **mercury** bound to human serum albumin in patients with **autism**. A significant percentage of children with **autism** developed anti-SK, anti-gliadin and casein peptides and anti-ethyl **mercury antibodies**, concomitant with the appearance of anti-CD26 and anti-CD69 autoantibodies. These **antibodies** are synthesized as a result of SK, gliadin, casein and ethyl **mercury** binding to CD26 and CD69, indicating that they are specific. Immune absorption demonstrated that only specific antigens, like CD26, were capable of significantly reducing serum anti-CD26 levels. However, for direct demonstration of SK, gliadin, casein and ethyl **mercury** to CD26 or CD69, microtiter wells were coated with CD26 or CD69 alone or in combination with SK, gliadin, casein or ethyl **mercury** and then reacted with enzyme labeled rabbit anti-CD26 or anti-CD69. Adding these molecules to CD26 or CD69 resulted in 28-86% inhibition of CD26 or CD69 binding to anti-CD26 or anti-CD69 **antibodies**. The highest % binding of these antigens or peptides to CD26 or CD69 was attributed to SK and the lowest to casein peptides. We, therefore, propose that bacterial antigens (SK), dietary peptides (gliadin, casein) and Thimerosal (ethyl **mercury**) in individuals with pre-disposing HLA molecules, bind to CD26 or CD69 and induce

antibodies against these molecules. In conclusion, this study is apparently the first to demonstrate that dietary peptides, bacterial toxins and xenobiotics bind to lymphocyte receptors and/or tissue enzymes, resulting in autoimmune reaction in children with **autism**.

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=> file .chemistry
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      11.35      11.56

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FILE 'USPATFULL' ENTERED AT 12:27:13 ON 18 MAY 2006
CA INDEXING COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

=> (lead or mercury) and antibody and (autism or autistic)
L9            7 FILE CAPLUS
L10           0 FILE BIOTECHNO
L11           0 FILE COMPENDEX
L12           0 FILE ANABSTR
L13           0 FILE CERAB
L14           0 FILE METADEX
L15         730 FILE USPATFULL

TOTAL FOR ALL FILES
L16         737 (LEAD OR MERCURY) AND ANTIBODY AND (AUTISM OR AUTISTIC)

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ENTER L# LIST OR (END):l9
PROCESSING COMPLETED FOR L9
L17           7 DUP REM L9 (0 DUPLICATES REMOVED)

=> d l17 ibib abs total

L17  ANSWER 1 OF 7  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2005:698214  CAPLUS
DOCUMENT NUMBER:          143:171341
TITLE:                    Methods for detecting infections, toxic chemicals and
                          dietary peptides binding to lymphocyte receptors and
                          tissue enzymes as instigators of autoimmunity in
                          autism
INVENTOR(S):              Vojdani, Aristo
PATENT ASSIGNEE(S):       USA
SOURCE:                   U.S. Pat. Appl. Publ., 89 pp.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
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PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005170333	A1	20050804	US 2004-770712	20040203
PRIORITY APPLN. INFO.:			US 2004-770712	20040203

AB The present invention provides methods for diagnosis and following up a prognosis of children with **autism** before and after treatment with different modalities administered by their clinicians, confirming the involvement of infectious agents, dietary proteins, and toxic chems. in development of **autism**. In particular, methods for detecting infections, toxic chems. and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in **autism** are described. The method utilizes detection of increased amts. of **antibodies** against an antigen based on infectious agent, toxic chems., or dietary proteins. Another method utilizes detection of **antibodies** to a self-tissue or peptide.

L17 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:452933 CAPLUS
DOCUMENT NUMBER: 141:37230
TITLE: Nuclear receptors as diagnostic and risk markers for disease and as targets for therapy
INVENTOR(S): Gaitanaris, George A.; Bergmann, John E.; Gracerov, Alexander; Hohmann, John; Li, Fusheng; Madisen, Linda; Mcilwain, Kellie L.; Pavlova, Maria N.; Vassilatis, Demetri; Zeng, Hongkui
PATENT ASSIGNEE(S): Nura, Inc., USA
SOURCE: PCT Int. Appl., 508 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004045369	A2	20040603	WO 2003-US36229	20031112
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003295500	A1	20040615	AU 2003-295500	20031112
PRIORITY APPLN. INFO.:			US 2002-426305P P	20021114
			WO 2003-US36229 W	20031112

AB Methods of using nuclear receptors as diagnostic markers for disease and for increased risk of disease and in the development of therapeutics for treatment of such diseases are described. The proteins and the genes encoding them may be used in diagnosis. Transgenic animals carrying the human genes for these receptors may be used in screening for effectors. The invention also provides methods for identifying compds. (e.g., agonists or antagonists) using the nuclear receptor polypeptides and polynucleotides of the invention, and for treating conditions associated with nuclear receptor dysfunction with the nuclear receptor polypeptides, polynucleotides, or identified compds. The invention also provides diagnostic assays for detecting diseases or disorders associated with inappropriate nuclear receptor activity or levels.

L17 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:674310 CAPLUS
DOCUMENT NUMBER: 142:22062
TITLE: Detection of Antinuclear and Antilaminin **Antibodies** in **Autistic** Children Who

Received Thimerosal-Containing Vaccines
AUTHOR(S): Singh, Vijendra K.; Rivas, Wyatt H.
CORPORATE SOURCE: Department of Biology, Utah State University, Logan, UT, USA
SOURCE: Journal of Biomedical Science (Basel, Switzerland) (2004), 11(5), 607-610
CODEN: JBCIEA; ISSN: 1021-7770
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Autism**, a neurodevelopmental disorder, may involve autoimmune pathogenesis. Since **mercury** is potentially a risk factor for autoimmunity, we conducted a study of **mercury**-induced antinuclear and antilaminin **antibodies** in **autistic** and normal children who had been pre-administered with thimerosal-containing vaccines. Laboratory anal. by different immunoassays showed that the serum level of these two autoimmune markers did not significantly differ between **autistic** and normal children. This finding suggests that the **mercury** as in thimerosal-containing vaccines is likely not related to autoimmune phenomenon in **autism**.
REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:649270 CAPLUS
DOCUMENT NUMBER: 140:89124
TITLE: Reduced Levels of **Mercury** in First Baby Haircuts of **Autistic** Children
AUTHOR(S): Holmes, Amy S.; Blaxill, Mark F.; Haley, Boyd E.
CORPORATE SOURCE: Baton Rouge, LA, USA
SOURCE: International Journal of Toxicology (2003), 22(4), 277-285
CODEN: IJTOFN; ISSN: 1091-5818
PUBLISHER: Taylor & Francis, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Reported rates of **autism** have increased sharply in the United States and the United Kingdom. One possible factor underlying these increases is increased exposure to **mercury** through thimerosal-containing vaccines, but vaccine exposures need to be evaluated in the context of cumulative exposures during gestation and early infancy. Differential rates of postnatal **mercury** elimination may explain why similar gestational and infant exposures produce variable neurol. effects. First baby haircut samples were obtained from 94 children diagnosed with **autism** using Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM IV) criteria and 45 age- and gender-matched controls. Information on diet, dental amalgam fillings, vaccine history, Rho D Ig administration, and **autism** symptom severity was collected through a maternal survey questionnaire and clin. observation. Hair **mercury** levels in the **autistic** group were 0.47 ppm vs. 3.63 ppm in controls, a significant difference. The mothers in the **autistic** group had significantly higher levels of **mercury** exposure through Rho D Ig injections and amalgam fillings than control mothers. Within the **autistic** group, hair **mercury** levels varied significantly across mildly, moderately, and severely **autistic** children, with mean group levels of 0.79, 0.46, and 0.21 ppm, resp. Hair **mercury** levels among controls were significantly correlated with the number of the mothers' amalgam fillings and their fish consumption as well as exposure to **mercury** through childhood vaccines, correlations that were absent in the **autistic** group. Hair excretion patterns among **autistic** infants were significantly reduced relative to control. These data cast doubt on the efficacy of traditional hair anal. as a measure of total **mercury** exposure in a subset of the population. In light of the biol. plausibility of **mercury**'s role in neurodevelopmental disorders, the present study provides further insight into one possible mechanism by which early **mercury** exposures could increase the risk of **autism**.
REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:44082 CAPLUS
DOCUMENT NUMBER: 140:216004
TITLE: Infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes are major instigators of autoimmunity in **autism**
AUTHOR(S): Vojdani, A.; Pangborn, J. B.; Vojdani, E.; Cooper, E. L.
CORPORATE SOURCE: Laboratory of Comparative Neuroimmunology, Department of Neurobiology, David Geffen School of Medicine at UCLA, University of California, Los Angeles, Los Angeles, CA, 90095, USA
SOURCE: International Journal of Immunopathology and Pharmacology (2003), 16(3), 189-199
CODEN: IJIPE4; ISSN: 0394-6320
PUBLISHER: Biolife s.a.s.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Similar to many complex autoimmune diseases, genetic and environmental factors including diet, infection and xenobiotics play a critical role in the development of **autism**. In this study, we postulated that infectious agent antigens such as streptokinase, dietary peptides (gliadin and casein) and Et **mercury** (xenobiotic) bind to different lymphocyte receptors and tissue enzyme (DPP IV or CD26). We assessed this hypothesis first by measuring IgG, IgM and IgA **antibodies** against CD26, CD69, streptokinase (SK), gliadin and casein peptides and against Et **mercury** bound to human serum albumin in patients with **autism**. A significant percentage of children with **autism** developed anti-SK, anti-gliadin and casein peptides and anti-Et **mercury antibodies**, concomitant with the appearance of anti-CD26 and anti-CD69 autoantibodies. These **antibodies** are synthesized as a result of SK, gliadin, casein and Et **mercury** binding to CD26 and CD69, indicating that they are specific. Immune absorption demonstrated that only specific antigens, like CD26, were capable of significantly reducing serum anti-CD26 levels. However, for direct demonstration of SK, gliadin, casein and Et **mercury** to CD26 or CD69, microtiter wells were coated with CD26 or CD69 alone or in combination with SK, gliadin, casein or Et **mercury** and then reacted with enzyme labeled rabbit anti-CD26 or anti-CD69. Adding these mols. to CD26 or CD69 resulted in 28-86% inhibition of CD26 or CD69 binding to anti-CD26 or anti-CD69 **antibodies**. The highest % binding of these antigens or peptides to CD26 or CD69 was attributed to SK and the lowest to casein peptides. We, therefore, propose that bacterial antigens (SK), dietary peptides (gliadin, casein) and Thimerosat (Et **mercury**) in individuals with pre-disposing HLA mols.; bind to CD26 or CD69 and induce **antibodies** against these mols. In conclusion, this study is apparently the first to demonstrate that dietary peptides, bacterial toxins and xenobiotics bind to lymphocyte receptors and/or tissue enzymes, resulting in autoimmune reaction in children with **autism**.
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:82298 CAPLUS
DOCUMENT NUMBER: 138:219855
TITLE: Vaccines, viruses, and voodoo
AUTHOR(S): Borchers, Andrea T.; Keen, Carl L.; Shoenfeld, Yehuda; Silva, Joseph, Jr.; Gershwin, M. Eric
CORPORATE SOURCE: Division of Rheumatology, Allergy and Clinical Immunology, University of California at Davis, Davis, CA, USA
SOURCE: Journal of Investigational Allergology and Clinical Immunology (2002), 12(3), 155-168
CODEN: JIAIEF; ISSN: 1018-9068
PUBLISHER: Hogrefe & Huber Publishers
DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Vaccinations are invaluable in protection from a wide variety of diseases that can cause substantial morbidity and mortality. Although a rare complication of vaccination, autoimmune disorders represent one of these morbidities. Recently, widespread public concern has arisen from case reports suggesting that-similar to what has been observed after natural viral infections-there might be an association between specific immunizations and autoimmune diseases. Herein we address the biol. plausibility of such a connection, focusing particularly on the examples of hepatitis B, rubella, and measles-mumps-rubella (MMR) vaccinations, and the autoimmune diseases they are potentially associated with. Our review of the available data suggests that, for the general population, the risk:benefit ratio is overwhelmingly in favor of vaccinations. However, the possibility cannot be ruled out that, in genetically susceptible individuals, vaccination can result in the unmasking of an autoimmune disease triggered by the immunization. We also critically examine the existing data suggesting a link between immunization against MMR and **autism**, and briefly discuss the controversial evidence pointing to a possible relationship between **mercury** exposure from vaccines and **autistic** disorders. There is a continued urgent need for rigorously designed and executed studies addressing these potential assocns., although the use of vaccinations remains a critical public health tool for protection against infectious disease.

REFERENCE COUNT: 119 THERE ARE 119 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:295400 CAPLUS

DOCUMENT NUMBER: 135:191427

TITLE: The neurotoxic etiology of the **autistic** spectrum disorders: a replication study

AUTHOR(S): Edelson, Stephen B.; Cantor, David

CORPORATE SOURCE: The Edelson Center for Environmental and Preventive Medicine, Inc., Atlanta, GA, 30342, USA

SOURCE: Toxicology and Industrial Health (2000), 16(6), 239-247
CODEN: TIHEEC; ISSN: 0748-2337

PUBLISHER: Arnold, Hodder Headline

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Although it has been recognized that **autism** is a disorder due to dysfunctional central nervous system functioning, a model that can account for the diversity of the symptoms in the syndrome and the concordant anomalies in metabolic functioning, in a sample of 20 **autistic** individuals, Edelson and Cantor demonstrated a body burden of neurotoxicants in over 90% of these individuals with 100% of these individuals demonstrating impaired liver detoxication processes. This current study examined an independent sample of 39 **autistic** individuals and was able to replicate the general findings of Edelson and Cantor. The authors further evidence the genetic and environmental aspects of this hypothetical process and believe the immune system injury secondary to the immunotoxins causes "activation" of the immune system leading to the production of autoantibodies against haptens (brain proteins attached to chemical mols.), and the subsequent damage as part of the process of neurotoxicity in the **autistic** spectrum. This process has as its final pathway one of free radical generation and mol. injury. This paper can not go into the complex details of this process at this time. All of the above **leads** to a spectrum of neurodevelopment dysfunction demonstrated as **autism**.

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> metal and (antigen or antibody) and (autism or autistic)

L18 3 FILE CAPLUS

L19 0 FILE BIOTECHNO

L20 0 FILE COMPENDEX

L21 0 FILE ANABSTR

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L22           0 FILE CERAB
L23           0 FILE METADEX
L24         571 FILE USPATFULL

TOTAL FOR ALL FILES
L25         574 METAL AND (ANTIGEN OR ANTIBODY) AND (AUTISM OR AUTISTIC)

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ENTER L# LIST OR (END):l18
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L26             3 DUP REM L18 (0 DUPLICATES REMOVED)

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L26  ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2004:452933  CAPLUS
DOCUMENT NUMBER:          141:37230
TITLE:                    Nuclear receptors as diagnostic and risk markers for
                          disease and as targets for therapy
INVENTOR(S):              Gaitanaris, George A.; Bergmann, John E.; Gracerov,
                          Alexander; Hohmann, John; Li, Fusheng; Madisen, Linda;
                          Mcilwain, Kellie L.; Pavlova, Maria N.; Vassilatis,
                          Demetri; Zeng, Hongkui
PATENT ASSIGNEE(S):       Nura, Inc., USA
SOURCE:                   PCT Int. Appl., 508 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
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     WO 2004045369         A2    20040603    WO 2003-US36229         20031112
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE,
             GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
             LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ,
             OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM,
             TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
             ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,
             TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     AU 2003295500         A1    20040615    AU 2003-295500          20031112
PRIORITY APPLN. INFO.:                     US 2002-426305P      P   20021114
                                           WO 2003-US36229      W   20031112
AB   Methods of using nuclear receptors as diagnostic markers for disease and
     for increased risk of disease and in the development of therapeutics for
     treatment of such diseases are described. The proteins and the genes
     encoding them may be used in diagnosis. Transgenic animals carrying the
     human genes for these receptors may be used in screening for effectors.
     The invention also provides methods for identifying compds. (e.g.,
     agonists or antagonists) using the nuclear receptor polypeptides and
     polynucleotides of the invention, and for treating conditions associated with
     nuclear receptor dysfunction with the nuclear receptor polypeptides,
     polynucleotides, or identified compds. The invention also provides
     diagnostic assays for detecting diseases or disorders associated with
     inappropriate nuclear receptor activity or levels.

L26  ANSWER 2 OF 3  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2004:905350  CAPLUS
DOCUMENT NUMBER:          141:370510
TITLE:                    Screening for agents modulating CIRL3-L (calcium
                          independent receptor of latrotoxin 3-like) protein
                          related activity and use for treating metal
                          disorders
INVENTOR(S):              Croll-Kalish, Susan; Torres, Richard; Murphy, Andrew
                          J.
```

PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 20 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2004213738	A1	20041028	US 2004-804532		20040319
PRIORITY APPLN. INFO.:			US 2003-459076P	P	20030331

AB Provided is a human Calcium Independent Receptor of Latrotoxin 3-Like (CIRL3-L) protein, as well as the encoding nucleic acid, methods for screening for agents capable of modulating CIRL3-L related activity and treating CIRL3-L-mediated conditions. Further provided are animal models useful for screening agents capable of ameliorating or reducing anxiety related disorders, obsessive-compulsive disorders, seizure related disorders and **autism** and other pervasive developmental disorders.

L26 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:391987 CAPLUS
DOCUMENT NUMBER: 136:395976
TITLE: System and method for assaying drugs effects on central nervous system
INVENTOR(S): Soreq, Hermona; Meshorer, Eran; Sklan, Ella; Shoham, Shai
PATENT ASSIGNEE(S): Yissum Research Development Company of the Hebrew University of Jerusalem, Israel
SOURCE: PCT Int. Appl., 114 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2002040994	A2	20020523	WO 2001-IL1051		20011114
WO 2002040994	A3	20021219			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2002023996	A5	20020527	AU 2002-23996		20011114
US 2004058357	A1	20040325	US 2003-432131		20030926
PRIORITY APPLN. INFO.:			US 2000-247970P	P	20001114
			WO 2001-IL1051	W	20011114

AB The invention relates to a method and system for evaluating an effect on the nervous system of a test drug by comparing the effect of such drug on AChE catalytic activity or isoform variance in the brain of a test animal following challenge by an AChE blocker (e.g. DFP) or a blocker of AChE and muscarinic receptors M1 and M2 (e.g. pyridostigmine) and comparing this effect with that of a known agent, preferably a non-selective muscarinic receptor blocker (e.g. scopolamine) or a specific M1 receptor blocker (e.g. pirenzepine). Also provided is a method of screening for a candidate drug that is a modulator of the expression of any one of AChE variants and isoforms by determining the effect of such drug on the translocation of an AChE isoform within a neuron. Further provided is a method of screening for a candidate drug aimed at affecting central nervous system properties which is a modulator of the interaction between AChE-R/RACK1/PKC.

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FILE 'PASCAL' ENTERED AT 12:36:18 ON 18 MAY 2006

=> toxic and antibody and (autism or autistic)
L27 0 FILE AGRICOLA
L28 0 FILE BIOTECHNO
L29 0 FILE CONFSCI
L30 0 FILE HEALSAFE
L31 0 FILE IMSDRUGCONF
L32 1 FILE LIFESCI
L33 0 FILE PASCAL

TOTAL FOR ALL FILES
L34 1 TOXIC AND ANTIBODY AND (AUTISM OR AUTISTIC)

=> d l34 ibib abs total

L34 ANSWER 1 OF 1 LIFESCI COPYRIGHT 2006 CSA on STN
ACCESSION NUMBER: 2004:108019 LIFESCI
TITLE: Infections, **toxic** chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes are major instigators of autoimmunity in **autism**
AUTHOR: Vojdani, A.; Pangborn, J.B.; Vojdani, E.; Cooper, E.L.
CORPORATE SOURCE: 8693 Wilshire Blvd., Ste. 200, Beverly Hills, CA 90211, USA; E-mail: DrAri@msn.com
SOURCE: International Journal of Immunopathology and Pharmacology [Int. J. Immunopathol. Pharmacol.], (20031200) vol. 16, no. 3, pp. 189-199.
ISSN: 0394-6320.
DOCUMENT TYPE: Journal
FILE SEGMENT: F
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Similar to many complex autoimmune diseases, genetic and environmental factors including diet, infection and xenobiotics play a critical role in the development of **autism**. In this study, we postulated that infectious agent antigens such as streptokinase, dietary peptides (gliadin and casein) and ethyl mercury (xenobiotic) bind to different lymphocyte receptors and tissue enzyme (DPP IV or CD26). We assessed this hypothesis first by measuring IgG, IgM and IgA **antibodies** against CD26, CD69, streptokinase (SK), gliadin and casein peptides and against ethyl mercury bound to human serum albumin in patients with **autism**. A significant percentage of children with **autism** developed anti-SK, anti-gliadin and casein peptides and anti-ethyl mercury **antibodies**, concomitant with the appearance of anti-CD26 and anti-CD69 autoantibodies. These **antibodies** are synthesized as a result of SK, gliadin, casein and ethyl mercury binding to CD26 and CD69, indicating that they are specific. Immune absorption demonstrated that only specific antigens, like CD26, were capable of significantly reducing serum anti-CD26 levels. However, for direct demonstration of SK, gliadin, casein and ethyl mercury to CD26 or CD69, microtiter wells were coated

with CD26 or CD69 alone or in combination with SK, gliadin, casein or ethyl mercury and then reacted with enzyme labeled rabbit anti-CD26 or anti-CD69. Adding these molecules to CD26 or CD69 resulted in 28-86% inhibition of CD26 or CD69 binding to anti-CD26 or anti-CD69 **antibodies**. The highest % binding of these antigens or peptides to CD26 or CD69 was attributed to SK and the lowest to casein peptides. We, therefore, propose that bacterial antigens (SK), dietary peptides (gliadin, casein) and Thimerosal (ethyl mercury) in individuals with pre-disposing HLA molecules, bind to CD26 or CD69 and induce **antibodies** against these molecules. In conclusion, this study is apparently the first to demonstrate that dietary peptides, bacterial toxins and xenobiotics bind to lymphocyte receptors and/or tissue enzymes, resulting in autoimmune reaction in children with **autism**.

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=> chemical and antibody and (autism or autistic)
L35           0 FILE AGRICOLA
L36           0 FILE BIOTECHNO
L37           0 FILE CONFSCI
L38           0 FILE HEALSAFE
L39           0 FILE IMSDRUGCONF
L40           1 FILE LIFESCI
L41           1 FILE PASCAL

TOTAL FOR ALL FILES
L42           2 CHEMICAL AND ANTIBODY AND (AUTISM OR AUTISTIC)

=> dup rem
ENTER L# LIST OR (END):l42
DUPLICATE IS NOT AVAILABLE IN 'IMSDRUGCONF'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L42
L43            2 DUP REM L42 (0 DUPLICATES REMOVED)

=> d l43 ibib abs total

L43  ANSWER 1 OF 2  LIFESCI    COPYRIGHT 2006 CSA on STN
ACCESSION NUMBER:   2004:108019  LIFESCI
TITLE:              Infections, toxic chemicals and dietary peptides
                    binding to lymphocyte receptors and tissue enzymes are
                    major instigators of autoimmunity in autism
AUTHOR:             Vojdani, A.; Pangborn, J.B.; Vojdani, E.; Cooper, E.L.
CORPORATE SOURCE:   8693 Wilshire Blvd., Ste. 200, Beverly Hills, CA 90211,
                    USA; E-mail: DrAri@msn.com
SOURCE:             International Journal of Immunopathology and Pharmacology
                    [Int. J. Immunopathol. Pharmacol.], (20031200) vol. 16, no.
                    3, pp. 189-199.
                    ISSN: 0394-6320.
DOCUMENT TYPE:      Journal
FILE SEGMENT:       F
LANGUAGE:           English
SUMMARY LANGUAGE:   English
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AB Similar to many complex autoimmune diseases, genetic and environmental factors including diet, infection and xenobiotics play a critical role in the development of **autism**. In this study, we postulated that infectious agent antigens such as streptokinase, dietary peptides (gliadin and casein) and ethyl mercury (xenobiotic) bind to different lymphocyte receptors and tissue enzyme (DPP IV or CD26). We assessed this hypothesis first by measuring IgG, IgM and IgA **antibodies** against CD26, CD69, streptokinase (SK), gliadin and casein peptides and against ethyl mercury bound to human serum albumin in patients with **autism**. A significant percentage of children with **autism** developed anti-SK, anti-gliadin and casein peptides and anti-ethyl mercury **antibodies**, concomitant with the appearance of anti-CD26 and anti-CD69 autoantibodies. These **antibodies** are synthesized as a result of SK, gliadin, casein and ethyl mercury binding to CD26 and CD69, indicating that they are specific. Immune absorption demonstrated that only specific antigens, like CD26, were capable of significantly reducing serum anti-CD26 levels. However, for direct demonstration of SK, gliadin,

casein and ethyl mercury to CD26 or CD69, microtiter wells were coated with CD26 or CD69 alone or in combination with SK, gliadin, casein or ethyl mercury and then reacted with enzyme labeled rabbit anti-CD26 or anti-CD69. Adding these molecules to CD26 or CD69 resulted in 28-86% inhibition of CD26 or CD69 binding to anti-CD26 or anti-CD69 **antibodies**. The highest % binding of these antigens or peptides to CD26 or CD69 was attributed to SK and the lowest to casein peptides. We, therefore, propose that bacterial antigens (SK), dietary peptides (gliadin, casein) and Thimerosal (ethyl mercury) in individuals with pre-disposing HLA molecules, bind to CD26 or CD69 and induce **antibodies** against these molecules. In conclusion, this study is apparently the first to demonstrate that dietary peptides, bacterial toxins and xenobiotics bind to lymphocyte receptors and/or tissue enzymes, resulting in autoimmune reaction in children with **autism**.

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ACCESSION NUMBER: 1995-0592064 PASCAL
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TITLE (IN ENGLISH): Low-dose naltrexone effects on plasma chemistries and clinical symptoms in **autism** : a double-blind, placebo-controlled study
AUTHOR: BOUVARD M. P.; LEBOYER M.; LAUNAY J.-M.; RECASENS C.; PLUMET M.-H.; WALLER-PEROTTE D.; TABUTEAU F.; BONDOUX D.; DUGAS M.; LENSING P.; PANKSEPP J.
CORPORATE SOURCE: Hop. Robert Debre, serv. psychopathologie enfant adolescent, 75019 Paris, France; Hop. Pitie Salpetriere, serv. psychiatrie adulte, 75013 Paris, France; Hop. Saint Louis, lab. neurochimie communications cellulaires, 75010 Paris, France
SOURCE: Psychiatry research, (1995), 58(3), 191-201, refs. 1 p.1/4
ISSN: 0165-1781 CODEN: PSRSDR
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: Ireland
LANGUAGE: English
AVAILABILITY: INIST-18303, 354000050442690020
AN 1995-0592064 PASCAL
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AB The effect of month-long naltrexone (NTX) treatment at a daily oral dose of 0.5 mg/kg/day was contrasted with placebo (PLC) in a double-blind study with conjoint clinical and biochemical evaluations of therapeutic effects. Modest clinical benefits were achieved with both PLC and NTX, with marginally better overall results following NTX, and degree of improvement appeared to be related to plasma **chemical** profiles. Massively elevated levels of β-endorphin were observed in all children with assays using C-terminal **antibody** but not with an N-terminal **antibody** assay. In addition, 70% of the children exhibited abnormally low levels of adrenocorticotropic hormone, and smaller subsets exhibited elevated norepinephrine (60%), arginine-vasopressin (50%), and serotonin (20%). The best clinical responders exhibited the clearest normalization of the elevated plasma chemistries, especially in C-terminal-β-endorphin and serotonin. There was some evidence of therapeutic carry-over effects in both clinical and biochemical measures in those children who received NTX before PLC. The results suggest that NTX only benefits a subgroup of **autistic** children, who may be identified by the presence of certain plasma abnormalities. These results suggest a possible linkage between abnormal plasma chemistries, especially those related to the pro-opiomelanocortin system, and **autistic** symptoms.

=> (PCB or xenobiotic or methylmercury) and antibody and (austism or autistic)
L44 0 FILE AGRICOLA
L45 0 FILE BIOTECHNO
L46 0 FILE CONFSCI
L47 0 FILE HEALSAFE

L48 0 FILE IMSDRUGCONF
L49 0 FILE LIFESCI
L50 0 FILE PASCAL

TOTAL FOR ALL FILES
L51 0 (PCB OR XENOBIOTIC OR METHYLMERCURY) AND ANTIBODY AND (AUSTISM OR AUTISTIC)

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NEWS 7	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 8	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
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